Reviews and Bibliographical Notices.

I.—THE VASO-MOTOR NERVOUS APPARATUS.

LECONS SUR L'APPAREIL VASO-MOTEUR (PHYSIOLOGIE ET PATHOLOGIE). Faites a la Faculté de Medecine de Paris. Par A. Vulpian. Redigées et Publiées par le Dr. H. C. Carville. Tome Second. Paris, 1875. P. 775. (*Lectures on the vaso-motor apparatus, etc.*)

In due time, the second volume of M. Vulpian's lectures on the *vaso-motor apparatus* has followed, and now we have the work complete.

During the past year we presented the readers of the JOURNAL with a pretty full review of the contents of the first, and now we proceed to notice in a similar manner the second and concluding volume.

At page 550 of the first volume, M. Vulpian begins the consideration of the vaso-motor nerves of the liver. He recites at length certain experiments performed by himself, which appear to show, that by irritation of the nerves of the hepatic plexus, which are conveyed to the liver on the vessels that enter its transverse fissure, the circulation in this organ can be modified in a remarkable manner, and still farther, that the pneumogastric nerves do not seem to be the channels either of vaso-constrictors or vaso-dilators. They are derived apparently from the cœliac ganglion, or in general terms, from the solar plexus, and certain ganglia which it includes. Everything, in fact, goes to show that wide fluctuations in blood-supply may be produced in the liver by influences won from the related vaso-motor nerves. Ever since the discovery of the glycogenic function of the liver by Bernard, and of the important part which the nervous apparatus of the liver plays in the same, its nervous supply has been, and yet is, an interesting question. And we desire before passing on to call the attention of our readers to the importance of a closer study of the structure, functions, and relations of the liver.

M. Vulpian now discusses the experimental results of Heidenhain, Rœhrig and Munk. The former, more particularly, opened the abdomen in dogs, and divided the common bile duct, and

adapted to the hepatic end of it a graduated tube in such way that the flow of bile could be estimated for given periods of time. These preliminary arrangements being completed, the spinal cord was excited in its cervical and dorsal portions, in different regions, and the effects on the flow of bile observed. At first the flow was increased, and then very much diminished. Upon ceasing the excitation of the spinal cord, the customary rapidity of the flow of the bile was slowly resumed. These phenomena were explained by Heidenhain, on the supposition that the irritation of the cord caused contraction of the bile ducts, and hence the flow was temporarily increased. But the consecutive diminished flow was supposed to depend on a contraction of the blood-vessels of the liver, thus diminishing the blood-supply to the organ, and hence the secretion and flow of the bile. But M. Vulpian questions the validity of this explanation, and then proceeds to develope his own views.

He found that division of the vagi nerves is followed by intense hyperæmia of the liver, and greatly increased secretion of the bile. Experimental lesions of the floor of the fourth ventricle also produce similar effects. How can these phenomena be explained? M. Vulpian does not think this question can be answered categorically, but is inclined to believe that in both cases certain secretory nerves of the liver are excited, by which means its secreting structure is increased in activity. But whatever the explanation of the phenomena may be they have a great pathological importance, for they show how the action of the liver may be profoundly modified by influences emanating from the nervous system.

But are there true secretory nerves for the liver? This seems to have been the most generally accepted view since the supposed discovery by Pflueger, that certain of the nerves of the liver terminate in the hepatic cells. But it must be remembered that the nerve terminations in the hepatic cells could not be established by Hering, though he employed the methods of Pflueger, and we are all the more inclined to reject the statements of the latter after reading the recent paper of Nesterowsky,* in which with new and improved methods, he was unable in a single instance to find a ganglion cell in the liver, or termination of a nerve fibre in a hepatic cell, as Pflueger claimed to have done previously. But notwithstanding these latest observations, and others earlier which seem to show that the secreting cells of the liver are devoid of nerve terminations, we are strongly inclined to admit their existence provisionally, not only in the liver, but in all secreting structures. The apparent discrepancy between the statement of M. Vulpian, that the pneumogastrics are without

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* *Ueber die Nerven der Leber.* Von Macarius Nesterowsky. Aus dem Laboratorium des Herrn Prof. Chrzonszcewsky. Eine von der medicinischen Facultaet der Universitaet Kiew gekroent Preisschrift. *Virchow's Archiv f. Path. Anat. u. f. Klin. Med.* Band 63, 3 u. 4 Heft. S. 412–421.

influence on the vessels of the liver, and that excitation or division of the vagus is followed by marked changes in the hepatic circulation, may be explained, perhaps, by what is said at another place in these volumes as to the source of the nerve fibres of the vagus. M. Vulpian says, on the basis of his own proper researches, that the vagus acquires at many points in its course new fibres, so that when it finally reaches its peripheral distribution it contains, out of the whole number of its fibres, not more than one in five which emerged from the cranial cavity with the pneumogastric. It is in its composition, in fact, a different nerve at its periphery, as compared with its more central course. The experiments of Bernard, who found that section of the pneumogastric above the diaphragm did not have the same influence on the functions of the liver as when made below it, confirm this view. It is the opinion of the latter that the pneumogastrics are the conductors of impression, probably from the lungs to the medulla oblongata, so as to excite a nervous mechanism in that region, which in its turn, in a reflex way influences the circulation in the liver, and perhaps modifies in an independent way the activity of its secreting structure. But the route of centrifugal impressions is, according to him, not in the pneumogastrics, though it may be and probably is in part, in the portion of the vagus which is near or below the diaphragm. But not only may lesions of the floor of the fourth ventricle give rise to modifications in the circulation and functions of the liver, but of other parts of the central nervous system, as the medulla itself (Bernard), the vermiform process of the cerebellum (Eckhard), or of the optic thalami, pons varolii, or middle peduncles of the cerebellum, or anterior columns of the spinal cord, in their whole length (Schiff). But injuries of none of these parts produce changes in the action of the liver with so much readiness as those in the floor of the fourth ventricle.

As to the nervous route from the floor of the fourth venticle, we believe, with M. Vulpian, that the opinion of Bernard is correct—that it is chiefly by the spinal cord and the splanchnic nerves, which latter terminate in the solar plexus, and then, last of all, by the nerves as already indicated which go from this plexus to accompany the vessels which enter the transverse fissure of the liver. And this nerve tract may be excited in either a direct or a reflex way.

M. Vulpian then passes to the discussion of the vaso-motor nerves of the heart, in doing which, he criticizes the view of Brown-Sequard, that the pneumogastrics contain nerves of this class. By this hypothesis, the latter has sought to explain the phenomenon of sudden arrest of the heart in diastole, which sometimes follows strong irritation of the pneumogastrics. The arrest, in his opinion, is produced by a sudden, perhaps a complete, contraction of the small arteries of the heart, through the assumed vaso-motor nerves of the vagi. The sudden anæmia of the muscular substance leads to its sudden inaction. But M.

Vulpian rejects this explanation, because, if the circulation in the small cardiac vessels is interrupted by injecting into them minute pellets of wax (Panum), or of lycopodium powder, or tobacco seed, and hence the passage of blood in them is interrupted, as perfectly as it is supposed to be in Brown-Sequard's hypothesis, yet the heart does not cease to act, as it does during excitation of the vagi. Moreover, tying of the coronary arteries does not cause the cessation of the heart's action, as should be, if the above-mentioned hypothesis is true. But still farther, direct observation of the heart, under strong excitation of the vagi nerves, fails to show any diminution in its vascularity; and then, again, the hearts of frogs, which are almost devoid of blood-vessels, are arrested by excitation of the vagi. For these various reasons M. Vulpian rejects the views of Brown-Sequard. How, then, does excitation of the vagi nerves arrest the heart's action? In all probability, by exerting a paralyzing influence on its intrinsic nervous apparatus. But we cannot follow this subject farther at present.

In the next place, the influence of the vaso-motor nerves on the vessels of the lungs is considered. Reference is made, of course, to the observations of Brown-Sequard on this subject (*Comptes Rendus de la Société de Biologie*, 1870, p. 86). He found that experimental lesions of the pons varolii are followed by congestions and hæmorrhages in the pulmonary tissue, at least quite often. By what channel are such effects produced? The channels of nervous influence are not the pneumogastrics, according to M. Vulpian, but the spinal cord, and then the various sympathetic filaments which pass off to the pulmonary plexuses, from the thoracic ganglia of the sympathetic, and then, to go farther backwards, the communicating branches between these last and the spinal cord. He adopts the opinion of Traube, that the appearance of congestion and œdema of the lungs after section of the vagi is really due to the entrance of food into the air passages, by reason of paralysis of certain of the muscles of the larynx. But M. Vulpian seems not to have noticed that this view has been disproved by the careful experiments of Genzmer,* who found that "hyperæmia and œdema of the lung arises after bilateral section of the vagus (its lung portion) without the introduction of any foreign substance into the lung," and "that vaso-motor nerves are contained in the pulmonary branches of the vagus, and that their paralysis produces hyperæmia of the lungs." But these conclusions of Genzmer are quite in accord with other observations of M. Vulpian himself, who has found, as we have already seen, that the vagus, as it descends the neck and thorax, acquires other nerve fibres at various points in its course, most of them probably vaso-motor nerves; but these conclusions are not

* *Gruende f. die Patholog. Veræderungen der Lungen, nach doppelseitige Vagusdurchschneidung.* Von Cand. Med. Alfred Genzmer. *Arch. f. d. gesammte Physiologie d. Menschen und Thiere.* 8 Band., s. 101-121.

in accord with those of our author, who holds that the vagus is without influence on the proper circulation of the lungs.

M. Vulpian now recounts certain experiments tending to show what influence the pneumogastrics have on the mucous membrane of the larynx. They seem to show that division and subsequent excitation of the central end of the pneumogastrics lead to a pronounced constriction of the vessels of the mucous membrane of the larynx. The vaso-motor nerves of these vessels therefore reach them by the route of the cervical sympathetic, rather than the pneumogastric proper. This fact leads to certain important practical deductions, which we cannot tarry in this place to notice.

In the next lecture the important subject of the "Vaso-motor Nerves of the Spinal Cord" is discussed with remarkable fullness and critical insight. The vessels with which the cerebro-spinal axis is so freely supplied are, like others, accompanied by vaso-motor nerves, both vaso-constrictors and vaso-dilators. M. Vulpian relates certain experiments which he has made on the vaso-motor nerves of the spinal cord, and with the following general results:

Excitation, by means of the induced current, of a communicating branch passing between a ganglion of the sympathetic and the root of a spinal nerve, led to constriction of the vessels that are visible on the surface of the cord, in that part of its course which corresponded to the irritated nerve fibres. If the excitation ceased, the vessels recovered their normal size, and if it was resumed they became again constricted. There could be no doubt of the fact. The same results followed excitation, by means of the induced current, of an intercostal nerve, between the point at which it receives a communicating branch from the sympathetic and that of its implantation in the spinal cord.

M. Vulpian now begins the discussion of the *reflex paralyses* of Brown-Sequard, with the intention of bringing to bear on them the light afforded by experiments such as we have just been referring to. We presume that it is known to the majority of our readers that Brown-Sequard, in the course of his numerous experiments on the kidneys and suprarenal capsules, noticed that if he ligated all the vessels and nerves of one or the other of these organs, the vessels of the pia mater of the spinal cord were at least temporarily contracted, and preferably, in the parts of the cord near the kidney, and the half of the cord which corresponded to the side of the wounded kidney. M. Vulpian admits having never performed the experiment, but sees no reason to doubt its correctness as reported. He draws from it, however, *en passant*, an argument in favor of the view which admits the existence of vaso-motor centres in the cord at all heights, as well as in the medulla. He says: "How can we explain the fact that the reflex contraction of the vessels provoked by the ligature of the nerves of the kidneys or of the suprarenal capsules, may be limited to a portion of the spinal cord so small in extent, and to

one side alone, if all the vaso-motor nerves of the body have their centre for reflex action in the medulla oblongata?"

But the chief point in the experiments of Brown-Sequard to which M. Vulpian would direct attention, is that they seem to prove that a vaso-constrictive reflex action provoked in the vessels of the pia mater of the cord by excitation of the nerves of a visceral organ (kidneys, for example), may not only be manifested in the pia mater, but the substance of the cord itself; and by reason of constriction of the vessels of the latter, to have for a consequence, at least theoretically, anæmia of this organ, and thus to abolish its action—producing, in fact, a more or less complete paralysis. Such a paralysis would be, in fact, succinctly stated, the "reflex paralysis" of Dr. Brown-Sequard. That anæmia, especially if complete, may lead to impairment, or even total abolition of the functions of the cord, has been abundantly established by experiment, as by Flourens, Panum, Pelz, and M. Vulpian himself; in all these cases, by immediate mechanical means—chiefly by artificial embolism.

Dr. Brown-Sequard used his experimental results just quoted, to explain the occurrence of certain paralyses which follow visceral disease, and which cases formerly passed under the name of "sympathetic paralyses." This latter physiologist supposed that, in case a paralysis followed on disease of the kidney or uterus, or bladder, etc., that it could be explained by supposing an excitation to pass along the centripetal nerves of the affected viscus to the spinal cord, and which was reflected out along vaso-motor nerves to the vessels of the cord, in such way as to cause their contraction, and hence, to deprive a portion of the cord of its blood-supply for varying periods in time, and in this way causing a paralysis, which he denominated "reflex."

The objections of Jaccoud and of Dr. S. Weir Mitchell to the views of Brown-Sequard, are discussed in a very exhaustive and critical manner, and are accepted, with certain qualifications, except as relates to the possibility of prolonged spasm of the muscular vessels, which is a cardinal point in the hypothesis of Dr. Brown-Sequard. The possibility of prolonged vasal spasm, so as by this means to produce the required anæmia as the immediate and sole condition of the paralysis, has been denied by M. Jaccoud, Dr. Mitchell and others. We have been for a long time of the opinion that this objection is not tenable, and we think there are but few who will read this able discussion of M. Vulpian who will feel inclined to maintain it.

Neither is M. Vulpian inclined to accept the theory of Jaccoud and Dr. Mitchell, viz.: of sudden exhaustion, by a kind of shock to the nervous centres. We cannot follow him into his elaborate criticism of the cases of reflex paralysis that have been reported by various authors, nor can we give more than a condensed statement of his own views. But he would range such cases among those in which a real structural lesion of the cord has been produced, even where none has been reported. Such lesions have

been found in many cases, and he believes them to exist in all. But he does not give such a place to ascending neuritis as it seems to us he should. The observations that have been collected recently under this head by careful writers like Friedreich (*Ueber progressive Muskelatrophie*, etc.) and Leyden (*Klinik d. Rueckenmarks Krankheiten*, B. II., Ersten Abth.), leave no doubt but that M. Vulpian is in error in so rigidly excluding it as an element in such cases, as he has done, more particularly in his introduction to the French translation of Dr. S. Weir Mitchell's admirable work on *Injuries to Nerves*, etc., and in which the subject of neuritis is treated at length, and in view of many new and valuable observations. There can be scarcely a doubt that inflammation may attack the nerves distributed to a viscus which is itself the seat of inflammatory disease, and extend from hence along the nerves in question to the spinal cord—in some instances very rapidly—and in this way kindle organic trouble in the cord at the point of their implantation, which may many times rapidly extend along the cord to break out with remarkable vigor in some remote and always higher part of the cerebro-spinal axis, giving rise in this way to marked symptoms, referable to parts of the cord not in immediate relation with the primitive seat of disease. Unless we admit some such mode of conveying morbid action from a viscus to the spinal cord, we do not see very clearly how M. Vulpian would explain the occurrence of lesions of the cord in certain cases that have been reported. That we may have a *neuritis migrans*, we think can hardly be doubted by any one who will read the evidence collected in an article published in the October number of this journal, by one of its editors. But while we are willing to consider an ascending neuritis, with consecutive alterations of corresponding parts of the spinal cord, as held by Friedreich, Leyden, Tiesler, Feinberg, Hayem, Weir Mitchell and others, as an established fact in certain cases, we are not among those who, like Friedreich, for example, hold it impossible to produce structural or trophic changes, either at the peripheral or central end of a nerve, by means of the so-called "nervous influence." We believe this to be possible, and can by no means admit, as yet, that in case of an apparent transmission of diseased action from a diseased viscus, to a nervous centre, or *vice versa*, that it must have been by a transmission of a neuritis, or some equivalent morbid structural change along the connecting nerve trunks.

In the next place, the *rôle* of the vaso-motor nerves in tetanus is considered, but this subject has been, to a certain extent, discussed in another part of this number of the JOURNAL, and is hence dismissed from present notice. Following this, are remarks on the *rôle* of the vaso-motors in hydrophobia, hysteria, and epilepsy. The experiments and hypotheses of Dr. Brown-Sequard, in respect to the latter disease, are very fully and clearly stated, and reference is made to the researches of Kussmaul and Tenner, Westphal and others, in respect to the same disease. The

question raised is, as to the mechanism through which the phenomena of the epileptic attack are produced.

M. Vulpian notices certain important differences in the results obtained by arresting the circulation in the carotids and the vertebral arteries. In the experiments he performed he used mostly the lycopodium powder, which being injected into the artery, is carried in the blood into the smaller arteries, which are in this way completely plugged, thus depriving the parts of blood by embolism. When the carotids are alone injected, loss of consciousness and of capacity for voluntary motion, occurs in a few minutes, but the respiratory and other actions, the nervous centres for the control of which are situated in the pons or medulla, are comparatively but little affected. But when the vertebral arteries are injected alone, the respiratory actions are much sooner involved than in the former case. But this is just what we should expect, *a priori*, when we consider the researches of Duret and Heubner, as to the regional distribution of the cranial arteries. The vertebral, supply the medulla and pons, as the cerebral do not. In this connection is noticed also, a difference in action as between chloroform, ether, chloral hydrate, etc., which lead rather to a suspension of consciousness and voluntary motion, and do not act until later on the respiratory centres; while nicotine has a reversed action, at least in the frog. Under this head, the important question is discussed as to what influence the vaso-motor nerves have on the vessels of the brain. The experiments of Bernard are quoted as to elevation of the temperature of the brain after section of the cervical sympathetic, and also those of Donders, Van der Beke Callenfels, and Nothnagel, as to the positive influence of the vaso-motors in the neck, on the size of the vessels of the brain, and the contradictory experiments of A. Schultz, Riegel and Jolly. He cites certain experiments of his own, that agree with those of the former observers, and which, in our judgment, represent the truth in the case, viz.: that there are fibres in the cervical sympathetic, and not there alone, which control the action of the muscular vessels of the brain, producing, according to varying circumstances, either contractions or dilatations of the same. In this connection M. Vulpian holds, in accordance with an opinion we have long entertained, that "all the vaso-motors destined to the vessels of the brain, are not contained in the cervical sympathetic. These vessels receive other vaso-motor nervous fibres, some of which are derived, it is true, from the superior cervical ganglion, while others are received from the extreme upper portions of the spinal cord, or even from the medulla oblongata, and which latter are not in relation with the superior cervical ganglion, and hence they may preserve their functions, as there is reason to think they do, after complete removal of the superior cervical ganglion of the sympathetic. It is therefore not surprising, that removal, or electrical excitation, either of the superior cervical ganglion or, with more reason, of the cervical sympathetic lower down,

should be followed by incomplete dilatation or contraction of the vessels of the brain." (Page 125, vol. II.) These views are confirmed by the experiments of Nothnagel, who observed contraction of the cerebral arteries to follow electrical excitation of the sensory nerves,—the crural, for example—notwithstanding the complete removal of the superior cervical ganglion of the same side. And the effect was more marked when he operated upon the peripheral expansions of the sensory nerves than when he operated on their trunks. The contradictory observations of Riegel and Jolly are explained by the fact that in their experiments, the animals were previously narcotized, as was not the case in those of Nothnagel.

M. Vulpian refers at some length, to the *epileptic zone*, discovered by Brown-Sequard in his experiments, more particularly on Guinea pigs, in the production of artificial epilepsy, and with which perhaps, the majority of our readers are already acquainted. This "epileptic zone" has its centre at the angle of the lower jaw and parts lying immediately about the same. Slight irritation of the skin within this region led to epileptic attacks, in the animals in which the epileptic state had been artificially generated. These results did not follow, at least with facility, irritation of the skin of any other part of the body. Section of the sensory nerves distributed to this region, rendered it impossible to provoke convulsive attacks, as was possible prior to the section of the nerves referred to. In respect to cerebral anæmia, as a cause of the loss of consciousness in epilepsy, M. Vulpian says of Guinea pigs, during the attacks of artificial epilepsy which have been excited in them, "I have examined directly the vessels of the membranous envelopes of the brain, and the color of the gray cortical substance of the cerebral hemispheres, during the epileptic attack in Guinea pigs, and I have never seen the intracranial vessels contract at the moment of the seizure." Not only so, but our author declares that he has never beheld a loss of consciousness follow severe electrical irritation of the cervical sympathetics, which causes, however, an immediate and very noticeable contraction of the arteries of the *pia mater*. But these experiments do not prove that sudden cerebral anæmia may not play a conspicuous part in the epileptic attack; because it is hard to decide, in many cases, as to a loss of consciousness in the lower animals, and in the case of electrical irritation of the cervical sympathetics, it has been already admitted that they contain only a small portion of the vaso-motors of the brain, and hence, operations conducted on these nerves must be to a certain extent without influence on the circulation in the brain. "Why," says M. Vulpian, "is it so strenuously maintained, that a contraction of the cerebral vessels plays so important a part in the epileptic attack? Is it not just to suppose that the anatomical elements themselves, in various parts of the brain, and which are involved in this attack, may be directly modified by special irritation coming from the periphery, or from

the nervous centres themselves? As for myself, I am greatly inclined to this view." In short, he doubts the correctness of the view which makes the epileptic attack depend chiefly on sudden anæmia produced by vasal spasm. As to the condition of the cerebral and cerebellar peduncles, the pons, the medulla, and upper part of the spinal cord, during the attack, he says it does not depend on either "an accumulation of blood in the medulla and base of the brain, nor on its richness in carbonic acid, nor yet on an arrest of the circulation in these parts." (P. 136.)

The views of M. Magnan are next discussed, who has been able to produce epileptoid attacks in certain of the lower animals by injecting *absinthe* into their veins in considerable quantities. During the period of the attack, M. Magnan has seen the cerebral vessels dilate rather than contract. Besides, he has seen the vessels in the fundus of the eye dilate during the attack in cases of epilepsy. But as regards the first case, M. Vulpian lays no stress, since independently of anæmia or hyperæmia, we have the presence and direct action of an irritant, which may immediately excite the anatomical elements of the brain and cord. In the second place he holds, with some show of propriety, that dilatation of the vessels of the fundus oculi is not sufficient proof that the cerebral vessels are in a similar state. What, then, is the opinion of M. Vulpian as to the mechanism of epilepsy? He has given utterance in this volume to no decided opinions, but is inclined to regard the order of phenomena in the attack to be about as follows: 1. Some irritative or exciting influence is conveyed to certain nervous centres. 2. A temporary paralysis or exhaustion of that part of the brain on which consciousness depends. 3. An excitation, in a reflex way, of the motor apparatus, which in turn leads to the convulsive movements. In other words, he believes that the vaso-motors play a subordinate part—if any—to say the most, in the mechanism of epilepsy. Both the changes in the calibre of the vessels, if such changes are admitted, and the convulsive action of the voluntary muscles, are declared to be truly reflex in character. As to those structural modifications of the nervous centres which may be transmitted hereditarily, but little is said, and that little is not only foreign to the scope of the work, but unsatisfactory.

But notwithstanding the prolonged and able discussion of M. Vulpian, we cannot share his conclusion as to the essential independence of the epileptic attack of sudden changes in vascularity of the nervous centres. But we cannot in this notice enter on a discussion of this topic. We have already done so in a brief manner in a former number of this journal. We have given so much space to a review of the foregoing points as they are treated by M. Vulpian, because they are intrinsically of the highest importance in their relations, both scientific and practical.

The next subject for consideration is the "theory of cerebral anæmia" in sleep. He rejects, and we think properly, alike the

theory of Durham, developed by Hammond, which makes sleep dependent on cerebral anæmia ; and the opposite theory of Cappie, in which it is held, that it depends on venous hyperæmia. He also would reject the theory which would make sleep dependent on the accumulation of acid, or other material, in the brain structure, as a result of excessive nervous activity and waste. Besides the theories just mentioned, M. Vulpian does not venture one of his own. In our judgment, no true theory of sleep has as yet been presented, and cannot rationally be, on any basis yet offered. In the next place M Vulpian details at length several experiments which were performed during the lecture before his hearers, in respect to the action of hypnotics on the intracranial circulation. His conclusion is that they are without any remarkable and no constant action on the vaso-motors of the nervous centres. He used especially, chloroform, ether, opium and chloral in these experiments, and relegates their effects either to direct action of hypnotic agents, or by a mechanism at present unknown.

The subject next in order is that of the "intra-muscular circulation." Under this head, reference is made to the researches which have been made under the direction of Ludwig, by a number of his students, the results of which chiefly appear in his "*Arbeiten.*" The sum of the researches referred to is, that the blood-current becomes more rapid in the vessels of the muscle during contraction than in the state of repose. This fact had also been noted and described, independently by Bernard, in his lectures on animal heat. The artificial excitation of the motor nerve leading to a muscle is followed by contraction of the related muscle, and, as already stated, by an increased flow of blood from the veins which lead away from the muscle, with two exceptions, namely : that if the muscle has been greatly fatigued immediately prior to the experiment, or if the animal had been previously curarized, the increased activity of the circulation did not appear. Not only is the flow of blood increased under the circumstances in question, but also the lymph. Now how can we explain the increased activity of the circulation of the blood in the muscle, during the state of contraction? In seeking an explanation of the phenomenon in question, M. Vulpian discusses at some length the results obtained by Hafiz—a student of Ludwig's—and rejects the results obtained in the Leipsic laboratory, and accepts those of Bernard. In short, electrization of the vaso-motor nerves going to the vessels of the muscles, caused their contraction, as in other organs, though in a less marked degree. Bernard found less carbonic acid and more oxygen in the venous blood which escapes from a muscle, the motor nerves of which had been divided, as compared with that from one, the motor nerves to which remained intact. Bernard explained this by assuming that, in the former case, the less amount of carbonic acid and the greater of oxygen in the venous blood were due to the fact of a loss of *tonus*, and hence of activity,

in the muscle, and hence less of a consumption of oxygen, and a corresponding diminished production of carbonic acid. In the latter case the conditions were changed because of the enduring *tonus*, or functional activity of the muscle. But M. Vulpian does not accept this explanation. He thinks the condition of the blood from the muscle, the motor nerves to which have been divided, is solely due to dilatation of the vessels of the muscle. But may it not be due to both these causes, especially the first? But to call up again the question already raised: By what mechanism is the circulation of the blood increased in a muscle during the state of contraction? M. Vulpian does not answer this question, though it is one of capital importance, in our judgment. We have tried to answer it in effect, but under different relations, in an earlier number of this journal, to which we would refer the reader. *

M. Vulpian now passes to a consideration of the influence of the vaso-motor nerves, on "animal heat." At this point the pathological part of the work professedly begins. To prepare the way for an intelligent treatment of his theme, he gives a brief history of researches bearing on it, from Lavoisier down to the present time. There can be no doubt that the chief sources of heat in the animal body, are the chemico-vital changes which take place in the blood and tissues, and between them, in those intimate processes of composition and decomposition which normally occur in the living body, or its tissues. The activities of the digestive system, of the secreting structures of all kinds, of muscle, of brain, are accompanied by the production of more or less heat, according as these parts or organs are more or less active. Now the real question is, as to what is the relation of the vaso-motor nerves to these heat-making processes, in the depths of the economy? In one way the vaso-motor nerves may, at least indirectly, influence the production of animal heat, viz.: by increasing or diminishing the volume of blood which circulates in a part in a given time; for it is largely owing to the oxygen that the blood conveys, that the changes occur on which the production of heat is immediately dependent. If there is more blood,—all other circumstances being equal—there will be more nutritive material and more oxygen, and hence the conditions for an increased production of heat, and *vice versa*. We have already seen that the calibre of the small muscular vessels is directly under the control of the vaso-motor nerves—constrictors and dilators. Hence, in a way that is quite manifest, the vaso-motors may influence the production of heat. M. Vulpian enters in the early part of his discussion of this question, into a peculiarly felicitous explanation of the relation of the cutaneous perspiration to the heat of the animal body, but inasmuch as it is not directly in the line of our subject, we pass it by. We would mention, however, in

*CHICAGO JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol I. p. 129, also p. 277.

passing, the explanation which is given of the fact that contraction of the cutaneous vessels and consequent paleness and coldness of the surface, is often followed by a notable increase in the temperature of parts in the interior of the body. It is highly satisfactory and is very suggestive, but we have not time nor space in which to refer to it in this notice.

M. Vulpian now passes to consider the subject of *fever*. An account is first given of the general phenomena of fever, and the etymology of the term, in which the remark is made that in these later times we have come to regard the essential feature in fever to be an abnormally high temperature. What is the mechanism for the augmentation of heat in fever? He states and then rejects the hypothesis of Traube, who held that the rise of temperature in fever is not due to an increased production of heat in the organism, but to a diminution in its loss. Also he rejects the theory of Marey, which is not unlike that of Traube with the exception that he does not explain the retention of heat in the same way as the latter. Next the views of Liebermeister and of Immermann are quoted, who were among the first to question and refute the hypothesis of Traube. They made certain definite observations which showed that the rise of temperature in fever could not be explained only in part by reason of a retention of heat. They also showed that there is augmented tissue change in fever, and of the kind which leads to the production of heat. This latter view has been essentially confirmed by a host of later observers. Now by what means is the increased tissue change produced? Is it due to an unusual supply of blood to the cutaneous surface of the body by reason of expansion of the small vessels, under influences won from the vaso-motor nervous system, or is it owing to certain irritated trophic nerves, which, by a direct action on the tissues to which they are distributed, increase the trophic or nutritive changes in the same, and without any necessary relation to vascular dilatation? As to the influence of the vaso-motors in causing contractions and dilatations of the small vessels, and thus by augmenting or diminishing the quantity of blood, there can be no longer any question. They play an important part, and in a way that is easily understood, in fever. Where there is less blood, there will be, all things being equal, less heat, and *vice versa*.

As to a direct influence of the nervous system on the intimate processes of nutrition, there can be no doubt of its existence, according to M. Vulpian. He enters on an elaborate statement of various researches bearing on this point, including those of Bernard, Keuchel, Heidenhain, Tscheschichin, Bruck and Guenther, Pochoy, Naunyn and Quincke, Riegel, Rosenthal and others, more particularly as respects a *moderator centre*. But a discussion of this subject is foreign alike to the scope of the work of M. Vulpian, and of this notice, both of which are professedly devoted to a consideration of vaso-motor, rather than *thermic* or *trophic* nerves. We may remark, however,

before passing, that our author feels justified, in view of all the facts that have come to his knowledge, in denying the existence of a heat-regulating *moderator centre* as located in the medulla or pons, or elsewhere, by various observers. This subject was, however, rather carefully discussed in an article in the July number of the JOURNAL for 1875, in a review of certain works of Senator, of Berlin, Winternitz, of Vienna, and of Dr. H. C. Wood, of Philadelphia.

(*To be Continued.*)

II.—LEYDEN : DISEASES OF THE SPINAL CORD.

KLINIK DER RUECKENMARKS KRANKHEITEN. Von Dr. E. Leyden, etc. I. Erster Band. Mit 8 zum Theil farbigen Tafeln. S. 478. II. Zweiter Band, Erste Abtheilung. Mit 6 zum Theil farbigen Tafeln. S. 300. Berlin : Hirschwald. (*Clinic on diseases of the spinal cord.*)

Since the appearance of the classical work of Ollivier (D'Angers), on *Diseases of the Spinal Cord*, nothing has been produced in medical literature that can compare with this of Dr. Leyden, which is likely to attain, when completed, a size of at least one thousand octavo pages. It is remarkable alike for its fullness of materials, both literary and original, for scientific thoroughness and practical character. It is written with great candor, and a certain kind of blunt independence, which inspires in the reader respect for its author, as well as for his work. Dr. Leyden is evidently no friend to speculation in medicine, and makes short work of its results, wherever he meets with them, in the writings of others. But it seems to us that he carries his realistic spirit too far, and in a resolute endeavor to avoid the dangers, misses all the palpable benefits arising from a rational and a cautious use of inference. The effects of this spirit are to be seen, not only in his methods, but in his style. The development of his theme is mechanical, rather than logical, and, as might have been expected, is devoid, in great measure, of that freedom in movement and suggestiveness, which a well regulated play of the imagination would have imparted to his work. But we only notice these points to make them the occasion for saying, that we hope to see the time when the present severely realistic spirit which pervades science will give way to one in which the true use of the imagination in the domain of inference will be understood, as it is not now.